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(FILE 'HOME' ENTERED AT 12:26:52 ON 06 OCT 2003)

FILE 'REGISTRY' ENTERED AT 12:31:29 ON 06 OCT 2003
L1 1 S SEPARASE/CN

FILE 'HCAPLUS' ENTERED AT 12:34:04 ON 06 OCT 2003

FILE 'REGISTRY' ENTERED AT 12:40:36 ON 06 OCT 2003
SET SMARTSELECT ON

L2 SEL L1 1- CHEM : 5 TERMS
SET SMARTSELECT OFF

FILE 'HCAPLUS' ENTERED AT 12:40:37 ON 06 OCT 2003

L3 71 S L2
L4 20 S L3 (L) (MAN OR HUMAN)
L5 8 S L4 AND PD<20010613
L6 3 S L5 AND INHIBIT?

=> d ibib ab 1-3

L6 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 2000:128329 HCAPLUS
DOCUMENT NUMBER: 132:261134
TITLE: Cell cycle mechanisms of sister chromatid separation;
roles of Cut1/separin and Cut2/securin
AUTHOR(S): Yanagida, Mitsuhiro
CORPORATE SOURCE: Department of Gene Mechanisms, Graduate School of
Biostudies, Kyoto University, Kyoto, 606-8502, Japan
SOURCE: Genes to Cells (2000), 5(1), 1-8
CODEN: GECEFL; ISSN: 1356-9597
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 30 refs. The correct transmission of chromosomes from mother to daughter cells is fundamental for genetic inheritance. Sepn. and segregation of sister chromatids in growing cells occurs in the cell cycle stage called "anaphase". The basic process of sister chromatid sepn. is similar in all eukaryotes: many gene products required are conserved. In this review, the roles of two proteins essential for the onset of anaphase in fission yeast, Cut2/securin and Cut1/**separin**, are discussed with regard to cell cycle regulation, and compared with the postulated roles of homologous proteins in other organisms. Securin, like mitotic cyclins, is the target of the anaphase promoting complex (APC)/cyclosome and is polyubiquitinated before destruction in a manner dependent upon the destruction sequence. The anaphase never occurs properly in the absence of securin destruction. In **human** cells, securin is an oncogene. **Separin** is a large protein (MW .apprx. 180 kDa), the C-terminus of which is conserved, and is thought to be **inhibited** by assocn. with securin at the nonconserved N-terminus. In the budding yeast, Esp1/**separin** is thought to be a component of proteolysis against Sccl, an essential subunit of cohesin which is thought to link duplicated sister chromatids up to the anaphase. Whether fission yeast Cut1/**separin** is also implicated in proteolysis of cohesin is discussed.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1999:485376 HCAPLUS
DOCUMENT NUMBER: 131:253894
TITLE: Identification of a vertebrate sister-chromatid separation **inhibitor** involved in transformation and tumorigenesis
AUTHOR(S): Zou, Hui; McGarry, Thomas J.; Bernal, Teresita; Kirschner, Marc W.
CORPORATE SOURCE: Department of Cell Biology, Harvard Medical School, Boston, MA, 02115, USA
SOURCE: Science (Washington, D. C.) (1999), 285(5426), 418-421
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A vertebrate securin (vSecurin) was identified on the basis of its biochem. analogy to the Pds1p protein of budding yeast and the Cut2p protein of fission yeast. The vSecurin protein bound to a vertebrate homolog of yeast **separins** Esp1p and Cut1p and was degraded by proteolysis mediated by an anaphase-promoting complex in a manner dependent on a destruction motif. Furthermore, expression of a stable Xenopus securin mutant protein blocked sister-chromatid sepn. but did not block the embryonic cell cycle. The vSecurin proteins share extensive sequence similarity with each other but show no sequence similarity to either of their yeast counterparts. **Human** securin is identical to the product of the gene called pituitary tumor-transforming gene (PTTG), which is overexpressed in some tumors and exhibits transforming activity in NIH 3T3 cells. The oncogenic nature of increased expression of vSecurin may result from chromosome gain or loss, produced by errors in

chromatid sepn.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1999:470065 HCAPLUS

DOCUMENT NUMBER: 131:240743

TITLE: Separating sister chromatids

AUTHOR(S): Nasmyth, Kim

CORPORATE SOURCE: IMP Research Institute of Molecular Pathology, Vienna,
A-1030, Austria

SOURCE: Trends in Biochemical Sciences (1999),
24(3), 98-104

CODEN: TBSCDB; ISSN: 0376-5067

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review, with .apprx.68 refs. Loss of cohesion between sister chromatids triggers their segregation during anaphase. Recent work has identified both a cohesin complex that holds sisters together and a sister-sepg. protein, **separin**, that destroys cohesion. **Separins** are bound by **inhibitory** proteins whose proteolysis at the metaphase-anaphase transition is mediated by the anaphase-promoting complex and its activator protein CDC20 (APCCDC20). When chromosomes are misaligned, a surveillance mechanism (checkpoint) blocks sister sepn. by **inhibiting** APCCDC20. Defects in this app. are implicated in causing aneuploidy in **human** cells.

REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT